

AMYGDALOID AND CORTICAL LESIONS
IN RATS: EFFECT ON RESPONSE TO
INCENTIVE CHANGE AND ON THE
LATENT EXTINCTION PHENOMENON

By
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CHAPTER I

INTRODUCTION

This study consisted of an investigation of the effect of lesions in the amygdaloid complex and in the lateral neocortex of laboratory rats on (1) changes in starting latency and running speed in a straight alley as a function of change in magnitude of food reward, (2) acquisition and latent extinction in a T maze, (3) change in threshold of reactivity or emotionality as determined by the King emotionality scale, and (4) the quantity of food consumed over two consecutive days.

The research had as its major aim the elucidation of a syndrome produced in monkeys by temporal lobectomy and described some years ago by Kluver and Bucy (1939). Included in the syndrome were changes in dietary behavior and changes in fear reactions and/or emotionality. Dietary anomalies took the form of indiscriminate feeding and a general failure to consider the palatability of objects, while the emotionality change tended to be in the direction of placidity and failure to avoid dangerous situations or noxious stimuli. A large number of studies have since indicated that most of the symptoms originally described by Kluver and Bucy have reference to structures located in the medial regions of the temporal lobe in or around the amygdaloid complex.

Secondly, the study had as a goal some clarification of the role of the amygdala as a structure within the limbic system, a system which has been viewed as playing a major role in insuring the continuity and sequencing of behavior (Pribram, 1960). This theoretical point of view, based in large part on observations of behavioral disruptions following lesions within the limbic system, provides a means of describing in broader perspective a number of seemingly unrelated behavioral changes produced by such lesions. Thus, the disturbance of hoarding and maternal behaviors observed in rats following cingulate lesions (Stamm, 1954; Stamm, 1955) could be described as reflecting the animal's inability to perform the component parts of a complex pattern of behavior in proper sequence. The disturbance of nest building activities in rats produced by cingulate lesions (Kim, 1960) might also point to the disruption of some sequencing mechanism. The reported observations of hyperphagia in amygdalectomized animals (Pribram and Bagshaw, 1953; Green, Clemente, and de Groot, 1957) is depicted as again reflecting a failure of sequencing--thus, "Feeding behavior may be difficult to initiate; once it has started it is difficult to stop" (Pribram, 1960). The sequencing concept, then, is generalized to include the initiation and termination of behavior at appropriate times.

Additional support for this theory comes through data taken from patients of the neurosurgeon (Milner, 1958). Resections of the amygdala

seemed to produce deficits in the patients' ability to carry out a prolonged digit span task in the presence of distracting stimuli and seemed to also negatively affect the persons' memory of how to get from one place to another--i.e., from the grocery store to his home. With these patients there appeared, however, to be no global loss of memory for events occurring prior to surgery. Such was not the case with a patient described by Terzian and Ore (1955). The patient, who had both temporal lobes removed, including most of the uncus and hippocampus, did not recognize his parents after the operations. Thus, the clinical data suggests that when the damage involves the hippocampus, there is a substantial loss of memory.

An alternative interpretation. Current speculation concerning the role of the limbic system in behavior has also developed along another theoretical line--i.e., it is central in the mediation of emotional expression and in the maintenance of adequate levels of motivation. Although the results of the majority of relevant studies have concurred with the observations of Kluver and Bucy that removal of the amygdala produces a marked lack of emotional responsiveness (Pribram and Bagshaw, 1953; Weiskrantz, 1956; King and Meyer, 1958), there have been some exceptions. Bard and Mountcastle (1948) reported an increase in aggressiveness of three amygdalectomized cats. Two of the three, however, sustained hippocampal damage. Rosvold, Mirsky, and Pribram (1954)

found that amygdalectomized monkeys responded aggressively toward their handlers, but less aggressively toward other monkeys. Schreiner and Kling (1953) produced several amygdalectomized cats with a low rage threshold using the same techniques which produced placidity in a number of others. Hence, the effect of amygdalectomy on emotionality does not seem to be entirely predictable.

In some cases where amygdalectomy has produced increased emotionality a focal discharge to the hippocampus may be responsible. Green (1964) has indicated that the hippocampus has a low seizure threshold in man and in animals and that injuries to Ammon's horn as well as electrical stimulation of this area can give rise to seizure discharges. Possibly conflicting results are also due in some cases to sub-total lesions producing non-standard effects (Wood, 1958), or, in other instances, to species differences. In addition, the way in which such terms as defense, emotionality, agonistic behavior, and aggressiveness tend to be used interchangeably but measured quite differently does very little to clarify the situation.

Electrical stimulation studies have also, in general, tended to add tenuous support to the contention that the amygdaloid complex is in some way associated with emotional reaction (MacLean and Delgado, 1953; Fondberg and Delgado, 1961; Wurtz and Olds, 1963; Hilton and Zybrozyna, 1963). Moreover, these studies concur in pointing to the phylogenetically

newer basolateral division of the amygdala as being more critical in such expression than is the older corticomedial portion. In addition, there is evidence that the amygdala modified aggressive behavior through its influence on the hypothalamus (Egger and Flynn, 1962; Kling and Hutt, 1958).

Negative and positive incentives. In learning experiments where shock has been used as a motivational variable with amygdalectomized Ss, the results have consistently indicated learning deficits (King, 1958; Weiskrantz and Wilson, 1958; Robinson, 1963). There is some indication, however, that the deficit may be positively related to the complexity of the task (Horvath, 1963). Since amygdalectomy does not significantly affect an animal's retention of an avoidance habit acquired prior to operation (Weiskrantz, 1956), the hypothesis, derived from clinical data, that the primary loss produced by such lesions is not a global loss of memory becomes even more tenable.

Whether amygdalectomized animals show a learning deficit when appetitive incentives are used has been found to depend greatly on the type of learning task. Delayed response learning, a task significantly affected by frontal lobe damage, seems to be unaffected by amygdaloid lesions (Pribram and Bagshaw, 1953). Furthermore, amygdalectomized animals show no deficit in learning simultaneous and successive visual and somesthetic discrimination problems (Schwartzbaum, 1965).

A study by Schwartzbaum (1960) utilizing an operant technique has indicated that amygdalectomized monkeys show a significant attenuation of response to changes in magnitude of food reward. Such a finding would seem to be quite compatible with either an earlier observation that amygdalectomy makes it difficult for animals to identify reinforcing stimuli (Weiskrantz, 1956) or that amygdalectomy reduces emotional responsiveness. Schwartzbaum appears to prefer the former alternative. Thus, he suggests that the attenuation reflects a loss of ability to discriminatively interrelate different reinforcing events rather than a specific dampening of emotional responsiveness.

Certainly some type of discrimination factor would have to be posited in interpreting the deficits shown by amygdalectomized animals in learning a transposition problem (Schwartzbaum and Pribram, 1960), a problem in stimulus equivalence transfer (Bagshaw and Pribram, 1965), and a learning set problem (Schwartzbaum and Poulos, 1965). Hence, amygdalectomy seems to impair processes that are involved in determining certain discriminative as well as reinforcing properties of stimuli.

Incentive-motivation. If we assume some motivational factor to be responsible for the observed attenuation of responsiveness of amygdalectomized animals to negative incentives and to shifts in positive incentives, the loss can be viewed in terms of an incentive-motivation factor.

According to Spence (1956), cues in an alley and goal box are conditioned to the goal response. Thus, the classically conditioned fractional anticipatory goal response tends to be elicited in the presence of these cues. Stimulus generalization, then, provides a mechanism by which the anticipatory goal response is evoked at points in the instrumental sequence prior to the goal response. The value of the incentive-motivation factor (K), which is presumed to have a multiplicative relationship with habit strength, is seen to be a function of the intensity or vigor of the anticipatory goal response. The intensity of this response is, in turn, a function of the number of reinforced trials, the delay of reinforcement, and the magnitude of reinforcement (Spence, 1960). The latter variable has especially been shown to be significantly related to level of performance in a straight runway (Crespi, 1942; Zeaman, 1949). Thus, runway performance with different reward magnitudes would seem to provide a reasonable test of any motivational changes produced by brain lesions. Moreover, a lack of responsiveness to shift in incentive magnitude can be viewed as reflecting a disruption of motivational as well as discriminative factors. If loss of incentive-motivation is responsible for attenuation of the amygdalectomized Ss' responsiveness to incentive shift, this motivational factor should also produce performance deficits prior to shift. Hence, attenuation of response to incentive shift requires interpretation in terms of pre-shift performance.

Latent extinction. Latent extinction, the counterpart of latent learning, refers to a reduction in response strength achieved by placing the animal in the goal box in the absence of reinforcement (Kimble, 1961). The phenomenon, as described by Kimble, reflects an extinction of the secondary reinforcing properties of stimuli in the goal box. Studies on latent extinction by Moltz (1955) and Moltz and Maddi (1956) suggest that this phenomenon is enhanced when the positive goal box is distinctive and readily discriminable from the non-positive one. If we assume that latent extinction reflects an extinction of the fractional anticipatory goal response, we might expect the process to be enhanced by distinctive goal box stimuli. The conditioning of the anticipatory response would appear to be maximal when goal box cues were distinctive, and, hence, the extinction of the response would be maximal when these distinctive cues were present without opportunity for the goal response to occur. Accordingly, a T maze experiment designed into the present study maximized the distinctiveness of the positive goal box.

Summary and hypotheses. The bilateral removal of the amygdaloid complex in a number of different species has produced similar behavioral changes for several of the species. However, contradictory and inconsistent findings have been frequent enough to warrant a further check on certain aspects of the "amygdaloid syndrome" and the manner in which it is related to other behavioral disturbances produced by

lesions in the limbic system.

Latency and running speed measures were obtained in a straight runway with two different magnitudes of reward. Incentive magnitudes were then shifted. Acquisition errors and latent extinction effects were recorded for T maze performance. A number of research hypotheses have been advanced for the runway and T maze experiments:

A. Ss with amygdaloid lesions are expected to demonstrate a significant attenuation of response to incentive shift in both latency and running speed. This prediction would seem to be reasonable in view of Schwartzbaum's results with amygdalectomized monkeys in an operant situation.

B. The attenuation will be significantly greater for latency than for running speed. Such a difference would be compatible with a suggestion that the threshold for initiating a sequence of behavior is high after amygdalectomy (Pribram, 1960).

C. Amygdalectomized Ss will be significantly less affected by latent extinction conditions than will the two control groups. However, the groups will not differ in terms of acquisition. If amygdaloid lesions selectively disrupt classical learning processes, and hence the conditioning of the fractional anticipatory goal response, the anticipatory response will then be relatively inoperative as a factor that could be reduced by latent extinction trials. Therefore, these Ss should show

significantly less latent extinction effect.

Since studies have not been in agreement concerning the effect of amygdalectomy on emotionality and food consumption, it would appear that no meaningful predictions can be made concerning these variables in the present study.

CHAPTER II

METHOD

Subjects. The Ss were 36 male, Long Evans rats approximately 100 days old at the time of surgery. They ranged in weight from 283 to 399 gm., with a mean of 335 gm. These animals, purchased from Research Animals Incorporated, were maintained on a 12 hr. light-dark cycle throughout the course of the experiments. They were placed on ad libitum food and water pre-operatively and remained on this schedule for 15 days post-operatively.

The animals were initially divided into three groups consisting of 12 animals per group. One group served as unoperated controls, another received lesions in the lateral neocortex, and bilateral lesions were made in the amygdaloid complex of animals in the third group.

Following surgery, five of the amygdalectomized animals had to be fed with a spoon and eyedropper. Four of the five began eating on the 3rd to 5th post-operative day, whereas the 5th animal was force fed until the 10th post-operative day at which time he died. Another animal in the operated control group developed a brain infection, failed to respond to antibiotic treatment, and died during the course of the experiments. Upon completion of all behavioral tests the operated animals were sacri-

ficed for histological examination.

Surgery. Interperitoneal injections of Nembutal were given based on a dosage of 60 mg. per kg. of body weight. This was then followed by a .12 mg. injection of atropine sulphate to reduce respiratory complications. The dorsal portion of the head was shaved and the external auditory meatus exposed prior to positioning in a Baltimore stereotaxic instrument. An anterior-posterior incision allowed access to the skull from which two lateral sections were removed with a trephine. The nose bar was placed 5 mm. above the interaural line, and four lesions were made bilaterally with a Grass R/F lesion maker. A current setting of 60 for 30 sec. was used. Coordinates for the amygdaloid lesions were (from bregma): anterior-posterior -.90, -.10, .70, and 1.50 mm.; lateral 4.75, 4.25, 4.00, and 4.00 mm.; ventral 9.30, 9.00, 9.00, and 8.75 mm. The scalp and ear incisions were stitched with silk suture, and approximately 24 hr. after surgery each animal was given a 60,000 unit intramuscular injection of procaine penicillin.

Apparatus. Running speed and latency were measured in a straight alley with a 15 in. entry box, a 12 in. start box, a 33 in. runway, and a 15 in. goal box. The flat black runway and goal box were 3 in. wide and 4 in. high. The entry and start boxes were 2 in. wide and painted flat gray. Both the alley and the boxes were covered with a 1/4 in. hardware cloth.

Latency and running time were measured electronically to 1/100 sec. by separate clocks activated respectively by a microswitch on the starting gate and a photoelectric cell positioned 3 in. in front of the start gate. The second clock was stopped by a microswitch located under the floor of the goal box.

The single unit T maze used to measure acquisition and latent extinction was modeled after one used by Moltz (1955). The stem was 21 by 5 in. and the top of the T was 24 by 5 in. Both were painted flat gray and were covered with a 1/4 in. hardware cloth. The goal boxes, painted flat black, were 10 by 10 in. square and were also covered with 1/4 in. hardware cloth. The entire T maze was covered with a triple thickness of cheese cloth suspended from a 4 by 4 ft. square frame to minimize the effect of extra-maze visual cues. A 25 watt light bulb was suspended directly over the center of the 4 ft. frame. A white melmac dish 6 in. in diameter was used as a food container for the acquisition and latent extinction series.

The King rating scale (1958) was used to measure the emotionality or reactivity of the animals. A sample copy of this scale is found in Appendix A.

Procedure. All animals were given emotionality ratings independently by two individuals for five days prior to operations and for three successive days following a two-week recovery period. Final emotion-

ality ratings were taken for three consecutive days following completion of the experiment on latent extinction. The ratings included, then, one pre-operative and two post-operative measures.

On the 11th post-operative day each animal was given a mixture of laboratory chow and water weighing approximately 200 gm. At the end of a 22 hr. period the dish and remaining food were again weighed and the difference between initial and second weights was recorded.

Seventeen days after operation all animals were placed on a 23 hr. food deprivation schedule on which they were maintained throughout the remainder of the experiments. Beginning on the 20th post-operative day the animals were given four trials daily for 12 consecutive days in the straight runway with either a 90 mg. or a 945 mg. food reward of 45 mg. Noyes pellets. At the end of 48 trials the animals receiving the small reward were shifted to the large and those receiving the large reward were shifted to the small. Following this shift in magnitude the animals were then given 24 additional trials in the runway at the rate of four per day. An intertrial interval of 2 min. was used for all trials. Starting latency and running time were measured electronically for each animal on each trial.

From 24 to 48 hr. after completion of the runway experiment all animals were given 10 trials per day in a T maze with a 180 mg. food reward of Noyes pellets for four consecutive days. Each goal box was

positive for approximately one-half the Ss in each group. A 10 min. intertrial interval was used. Approximately 24 hr. after completion of the acquisition series five animals from each of the three groups were assigned to a normal extinction group and six from each of the three groups to a latent extinction condition. One animal from the unoperated control group was inadvertently omitted from the latent extinction experiment. Animals in the latent extinction group were placed in the goal box with the empty food dish for four 1-min. periods separated by 10 min. intertrial intervals. Immediately following latent extinction the Ss were run with a 10 min. intertrial interval to an extinction criterion of two errors in any block of four trials. Animals in the normal extinction group which were not placed in the goal box were run to the same criterion using the same intertrial interval. The extinction score for each animal was the number of correct responses made before reaching the extinction criterion.

Histology. The animals were perfused with isotonic saline followed by a 10 per cent formalin solution. Their brains were removed, embedded in celoidin, and sectioned at 20 micra. Every 5th section was placed on a slide and stained with cresyl violet. The location and extent of lesions was reproduced on standard rat brain sections which were then photographed (see Appendix B).

CHAPTER III

RESULTS

Runway performance. Latencies and running time measures were converted to reciprocals. Hence, the graphs and analyses are based on reciprocal values. Latency measures obtained with a 945 mg. pre-shift reward and a 90 mg. post-shift reward are summarized in Figure 1. The mean values are given for consecutive series of four trials daily. An analysis of variance of the last pre-shift and the 1st post-shift sessions, presented in Table 1, indicates no significant differences among groups. The incentive-shift effects and the interaction, however, are statistically significant.

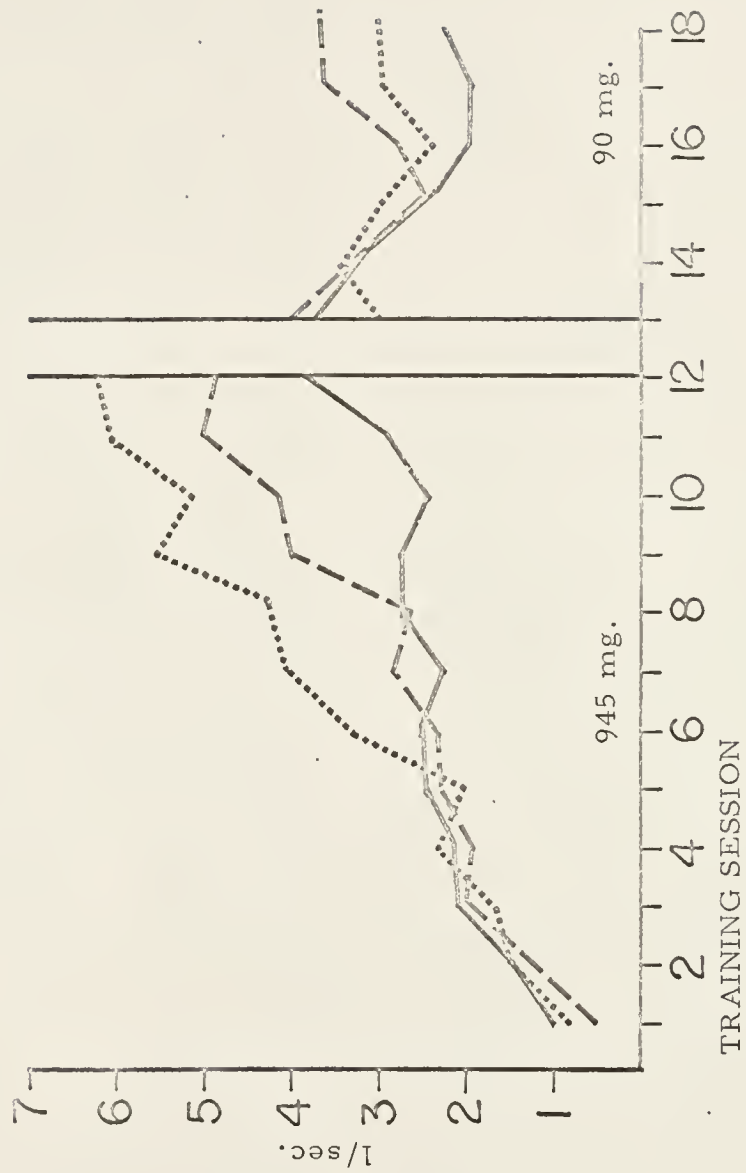


Figure 1. Means of reciprocals of starting latency in a straight alley with shift to small incentive.

TABLE 1

ANALYSIS OF VARIANCE OF LATENCY FOR LAST PRE-SHIFT
SESSION WITH 945 MG. REWARD AND 1ST POST-SHIFT
SESSION WITH 90 MG. REWARD

Source	d. f.	M. S.	F	P
Between <u>Ss</u>	17			
Groups	2	2.9192	.862	
Error between <u>Ss</u>	15	3.3859		
Within <u>Ss</u>	18			
Incentive-shift	1	19.5069	10.541	.01<
Incentive-shift X groups	2	7.1070	3.840	.05<
Error within <u>Ss</u>	15	1.8506		
Total	35			

Figure 2 shows reciprocal latency with training on a 90 mg. reward schedule and shift to 945 mg. An analysis of variance (Table 2) indicates that the groups effect, the incentive-shift effect, and the interaction are significant.

TABLE 2

ANALYSIS OF VARIANCE OF LATENCY FOR LAST PRE-SHIFT
SESSION WITH 90 MG. REWARD AND 1ST POST-SHIFT
SESSION WITH 945 MG. REWARD

Source	d. f.	M. S.	F	P
Between <u>Ss</u>	15			
Groups	2	7.7793	3.2872	.05<
Error between <u>Ss</u>	13	2.3665		
Within <u>Ss</u>	16			
Reward magnitude	1	26.7729	31.3941	.001<
Reward X groups	2	3.3505	3.9288	.01<
Error within <u>Ss</u>	13	.8525		
Total	31			

Means for reciprocals of running time with training on a 945 mg. reward and shift to 90 mg. are plotted in Figure 3. An analysis of variance (Table 3) indicates significance for the incentive-shift and the groups effect. Figure 4 shows running speed with a shift up in magnitude of reward. The analysis of variance (Table 4) indicates no significant group or interaction effect. It does, however, indicate significance for the incentive-shift factor.

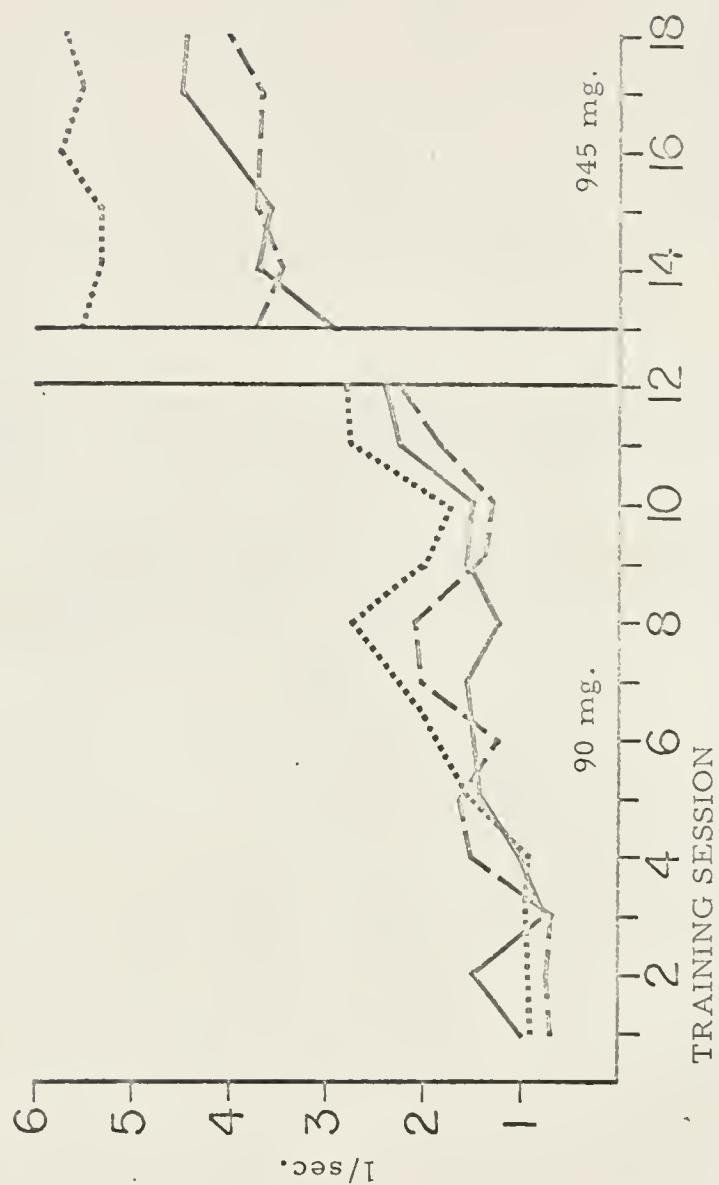


Figure 2. Means of reciprocals of starting latency in a straight alley with shift to large incentive.

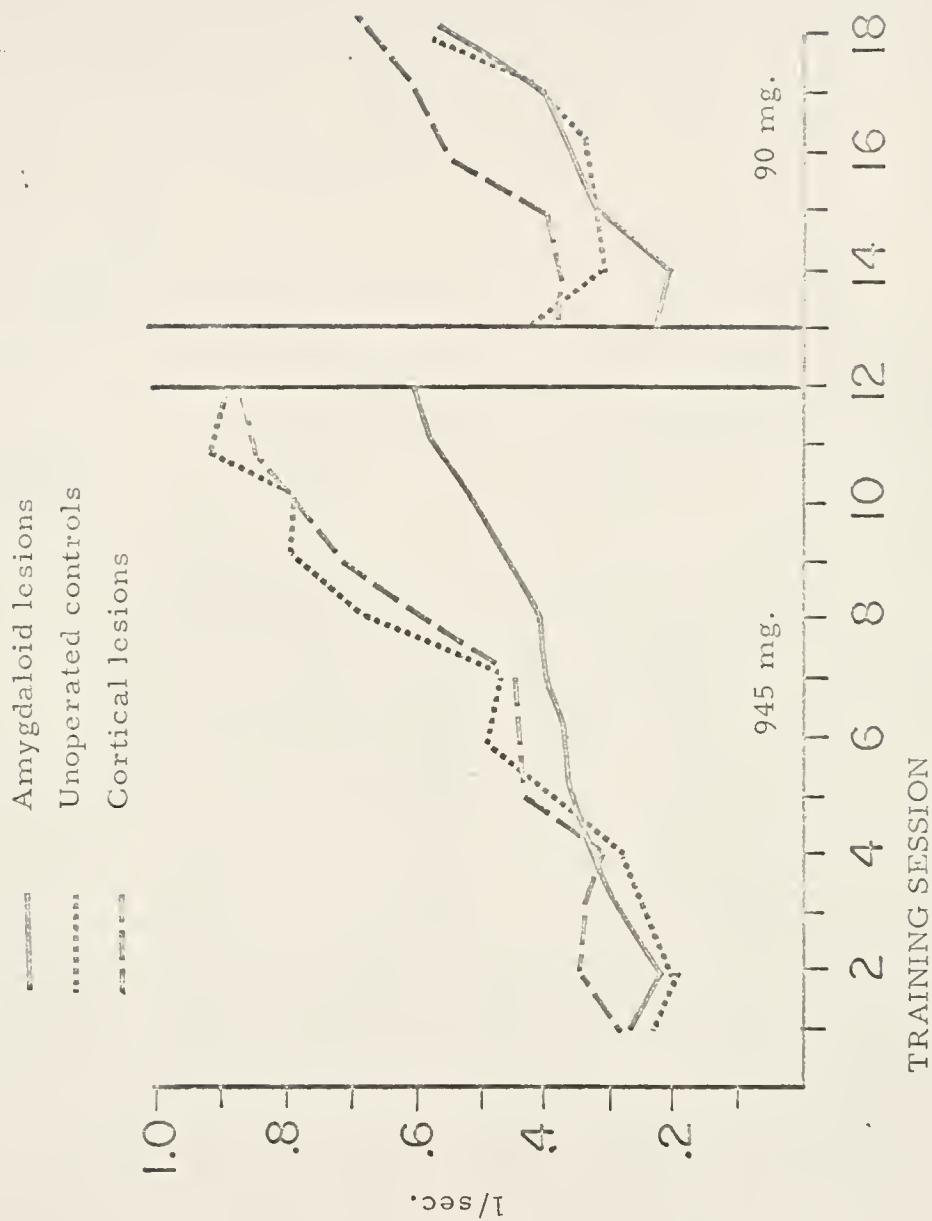


Figure 3. Means of reciprocals of running time in a straight alley with shift to small incentive.

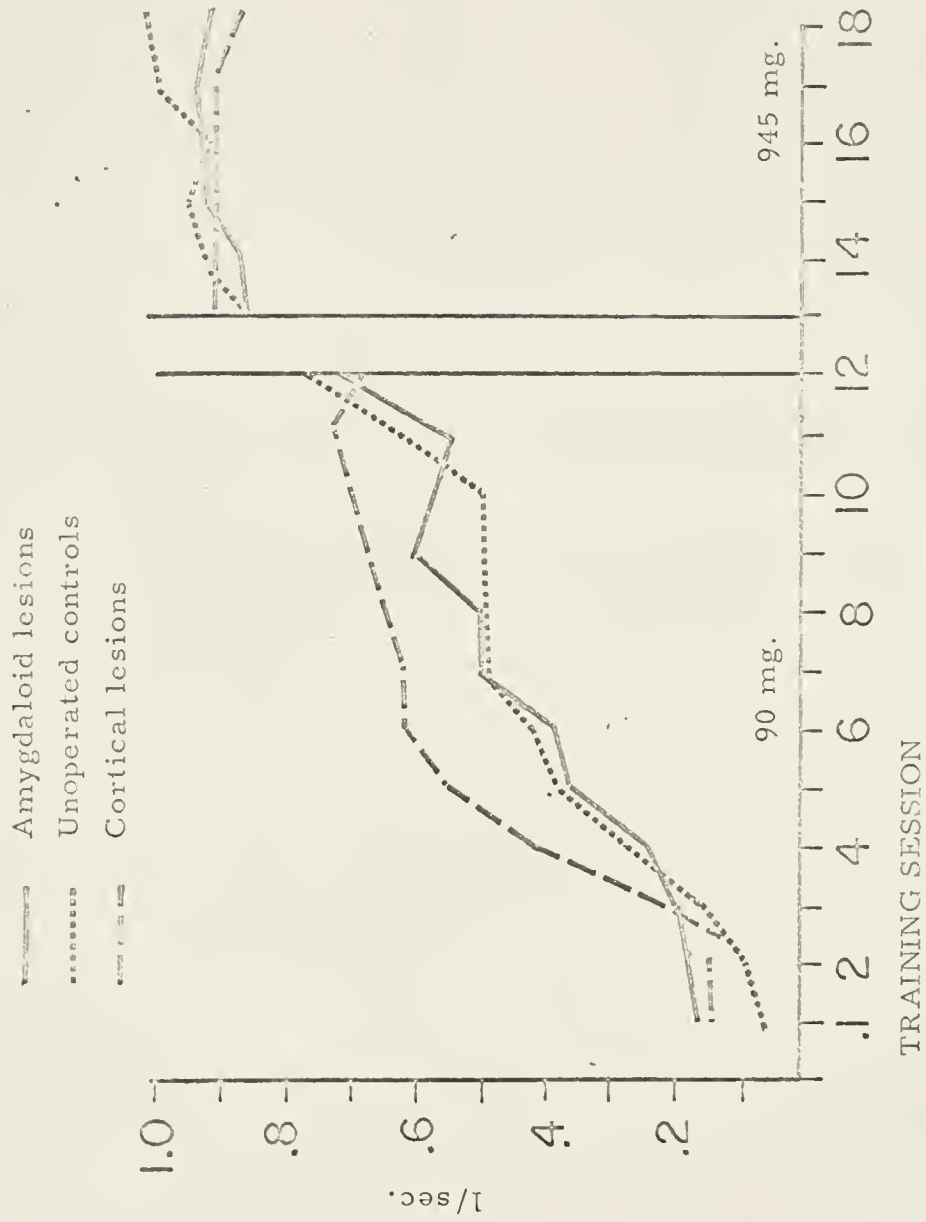


Figure 4. Means of reciprocals of running time in a straight alley with shift to large incentive.

TABLE 3

ANALYSIS OF VARIANCE OF RUNNING SPEED FOR LAST
PRE-SHIFT SESSION WITH 945 MG. REWARD AND
1ST POST-SHIFT SESSION WITH 90 MG. REWARD

Source	d.f.	M.S.	F	P
Between <u>Ss</u>	17			
Groups	2	.286957	12.0879	.01<
Error between <u>Ss</u>	15	.023739		
Within <u>Ss</u>	18			
Incentive-shift	1	1.476225	39.5362	.001<
Incentive-shift X groups	2	.016919	.4525	
Error within <u>Ss</u>	15	.037385		
Total	35			

An analysis of reward by groups for the last pre-shift session shows a significant reward effect for latency (Table 5) and a significant interaction for running speed (Table 6). The group effect for latency approaches but does not attain statistical significance.

Running speed and latency by trials for the 1st post-shift session are presented in Figure 5. Since the data on running speed with a 90 mg. reward seemed to offer the best possibility of a significant interaction, an *F* test was computed for this data. Although the intrasession effect did turn out to be significant, the interaction was not significant (Table 7).

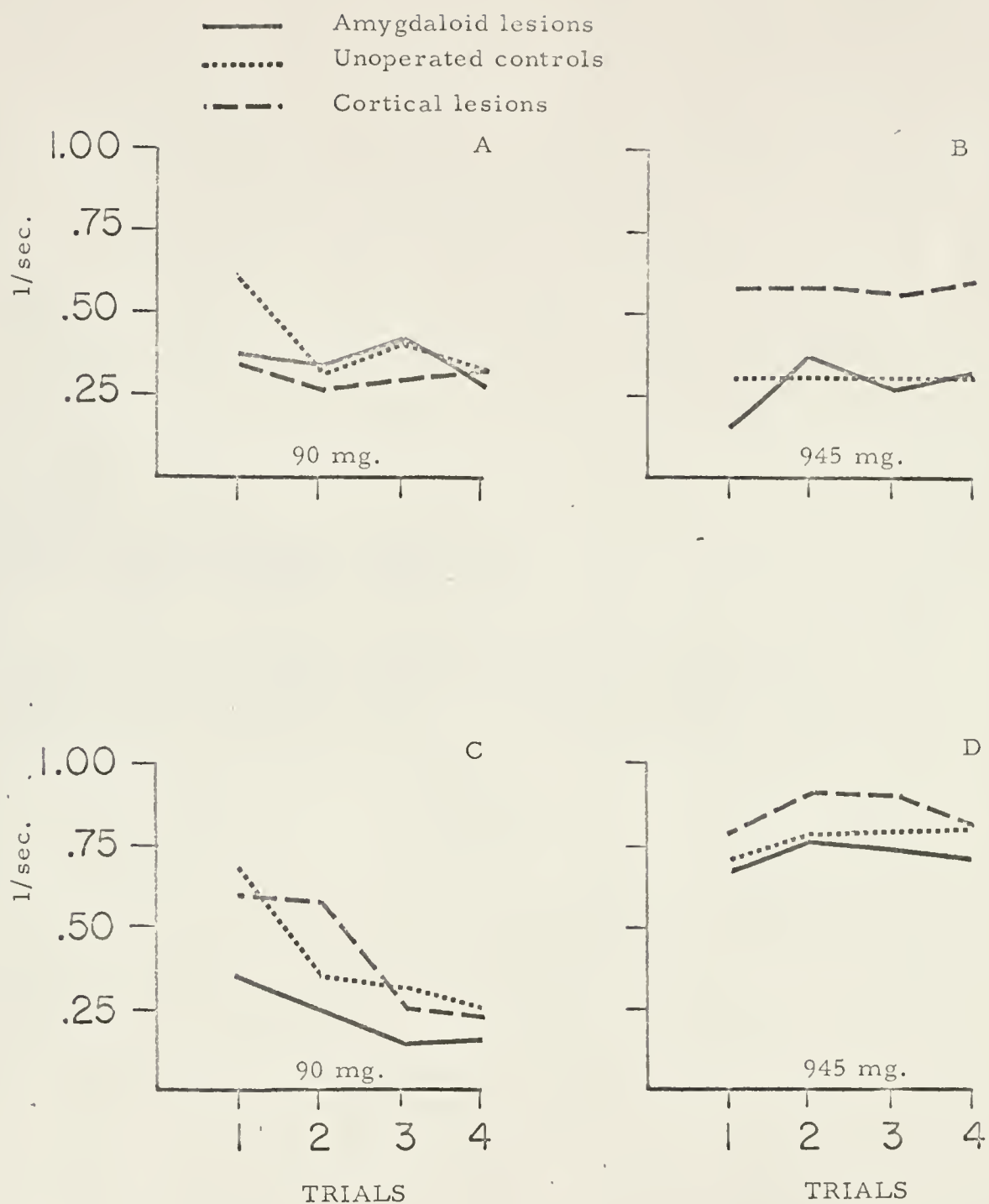


Figure 5. Means of reciprocals of latency trials (A and B) and running time (C and D) for 1st post-shift session.

TABLE 4

ANALYSIS OF VARIANCE OF RUNNING SPEED FOR LAST
PRE-SHIFT SESSION WITH 90 MG. REWARD AND
1ST POST-SHIFT SESSION WITH 945 MG. REWARD

Source	d. f.	M. S.	F	P
Between <u>Ss</u>	15			
Groups	2	.007481	.3188	
Error between <u>Ss</u>	13	.023461		
Within <u>Ss</u>	16			
Incentive-shift	1	.291848	48.4475	.001<
Incentive-shift X groups	2	.003244	.5385	
Error within <u>Ss</u>	13	.006024		
Total	31			

TABLE 5

ANALYSIS OF VARIANCE OF DIFFERENCE SCORES
OF RUNNING SPEED FOR 6TH AND 1ST POST-SHIFT
SESSIONS WITH 90 MG. REWARD

Source	d. f.	M. S.	F	P
Between groups	2	.2330	3.9897	.05<
Within groups	15	.0584		
Total	17			

TABLE 6

ANALYSIS OF VARIANCE OF DIFFERENCE SCORES
OF RUNNING SPEED FOR 6TH AND 1ST POST-SHIFT
SESSIONS WITH 945 MG. REWARD

Source	d. f.	M. S.	F	P
Between groups	2	.0627	3.7321	.05<
Within groups	13	.0168		
Total	15			

TABLE 7

ANALYSIS OF VARIANCE OF LATENCY FOR 6TH AND 1ST
POST-SHIFT SESSIONS WITH 945 MG. REWARD

Source	d. f.	M. S.	F	P
Between <u>Ss</u>	15			
Groups	2	13.9736	6.1978	.05<
Error between <u>Ss</u>	13	2.2546		
Within <u>Ss</u>	16			
Session	1	1.9801	2.9501	
Session X groups	2	1.2053	1.7957	
Error within <u>Ss</u>	13	.6712		
Total	31			

TABLE 8

RANDOM GROUPS ANALYSIS OF VARIANCE OF LATENCY
FOR LAST PRE-SHIFT SESSION

Source	d. f.	M. S.	F	P
Reward magnitude	1	63.54	33.44	.001 <
Group	2	6.26	3.29	
Reward X group	2	2.87	1.40	
Error	28	1.90		
Total	33			

TABLE 9

RANDOM GROUPS ANALYSIS OF VARIANCE OF RUNNING
SPEED FOR LAST PRE-SHIFT SESSION

Source	d. f.	M. S.	F	P
Reward magnitude	1	.092011	.6054	
Group	2	.068959	.4537	
Reward X group	2	1.507090	9.6170	.01 <
Error	28	.156980		
Total	33			

TABLE 10

ANALYSIS OF VARIANCE OF INTRASESSION EFFECTS ON RUNNING SPEED WITH 90 MG. REWARD FOR 1ST POST-SHIFT SESSION

Source	d. f.	M. S.	F	P
Trial	3	.401296	5.140	.01 <
Groups	2	.261097	3.345	.05 <
Trials X groups	6	.043250	.554	
Error	60	.078064		
Total	71			

T maze and latent extinction. A random analysis of variance for the number of errors made during the 40 acquisition trials on the T maze shows no significant group differences. Although inspection of the extinction data suggests that the amygdalectomized animals might be less responsive than the other groups to the effects of latent extinction (Table 11), an F test indicates no significant main effects or interaction. (M.S. error of 7.503, d.f. 30 for acquisition; M.S. error of 9.156, d.f. 27 for extinction.)

TABLE 11

MEAN NUMBER OF CORRECT RESPONSES OCCURRING
ON T MAZE IN ACQUISITION AND UNDER NORMAL
AND LATENT EXTINCTION CONDITIONS

	Acquisition	Latent Extinction	Normal Extinction
Amygdaloid Lesions	32.18	6.67	6.80
Cortical Lesions	33.55	5.83	8.60
Unoperated Controls	32.55	5.83	8.40

Emotionality. Separate analyses were done on difference scores between the pre-operative and 1st post-operative emotionality ratings (Table 12) and between the pre-operative and 2nd post-operative ratings (Table 13). These F tests indicated that there were significant differences among emotionality scores for the three groups as shown in Figure 6.

TABLE 12

ANALYSIS OF VARIANCE OF DIFFERENCE IN EMOTIONALITY
SCORES BETWEEN PRE-OPERATIVE AND 1ST POST-
OPERATIVE RATINGS

Source	d.f.	M.S.	F	P
Between groups	2	11.257	6.217	.01<
Within groups	30	1.811		
Total	32			

TABLE 13

ANALYSIS OF VARIANCE OF DIFFERENCE IN EMOTIONALITY
SCORES BETWEEN PRE-OPERATIVE AND 2ND POST-
OPERATIVE RATINGS

Source	d.f.	M.S.	F	P
Between groups	2	10.5025	4.171	.05<
Within groups	31	2.5180		
Total	33			

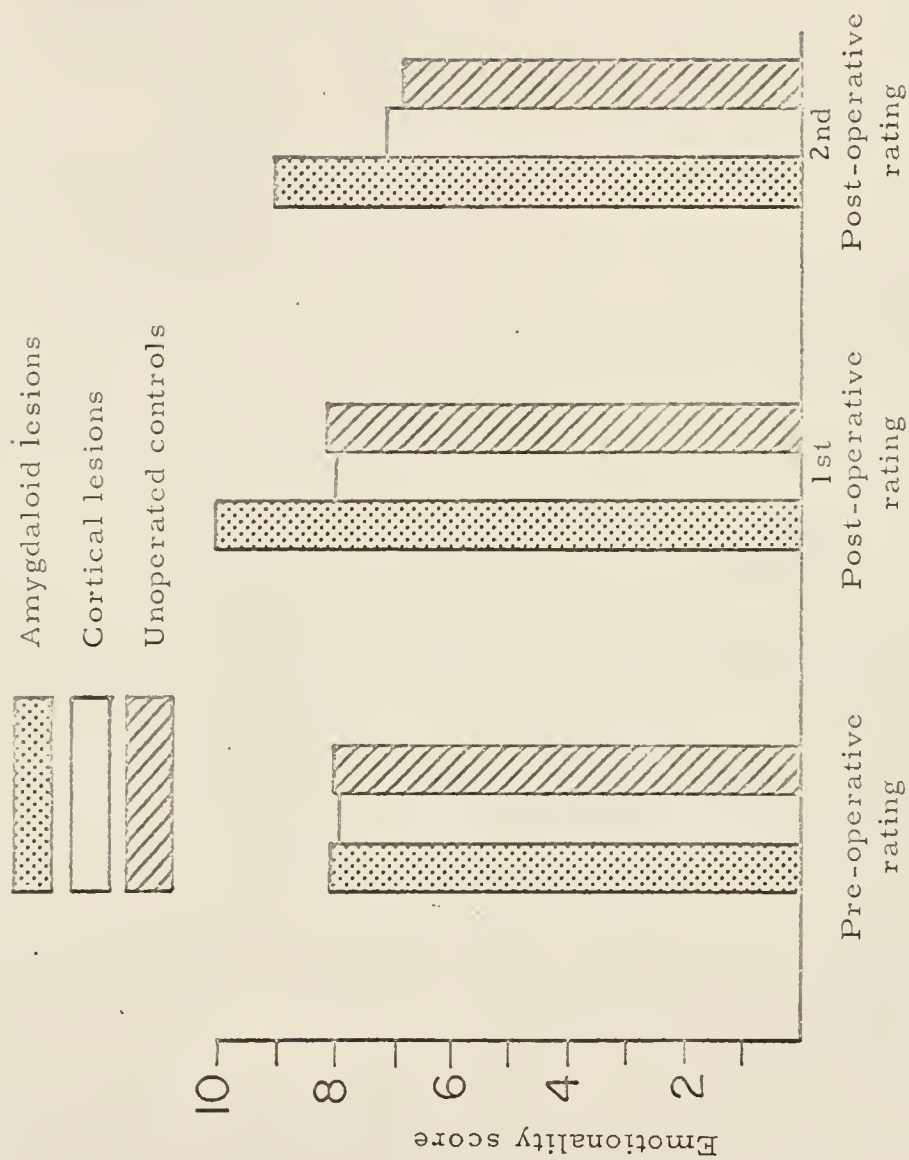


Figure 6. Emotionality scores for pre-operative and 1st and 2nd post-operative ratings.

A rank order correlation of .25 between posterior extent of amygdaloid lesions and the 2nd post-operative emotionality scores is not significant. An interrater reliability coefficient of .96 for emotionality ratings was significant ($P < .01$).

Food consumption. Analysis of the data on food consumption indicates that the three groups did not differ significantly on this variable. In fact, the amygdalectomized animals and the operated controls had almost identical means of 45.03 gm. and 45.76 gm. for the amount consumed over the two-day period.

Data summary. 1) Statistical tests of the effect of shift in magnitude of reward on latency and running speed show a significant groups by incentive-shift interaction for latency under both the shift up and shift down conditions. The incentive-shift by groups interaction for running speed, however, is not significant for either shift condition. In all four cases the incentive-shift effects are significant, but group differences were significant only for latency with a shift from small to large reward and for running speed with a shift from large to small reward. Differences between the 6th and 1st post-shift sessions for running speed indicated significant between groups effects for both the 90 mg. and the 945 mg. reward conditions. Within groups F tests of reciprocal latency measures for the 1st and 6th post-shift sessions indicated a significant between groups effect only for the 945 mg. reward condition. Pre-shift

data shows a significant reward effect on latency and a significant reward by groups interaction for running speed. Post-shift intrasession and group effects were significant for running speed under the small reward condition, but there was no significant trials by groups interaction.

2) There were no statistically significant differences between groups in terms of acquisition or extinction conditions on the latent extinction problem in the T maze, although the amygdalectomized Ss tended to extinguish more rapidly under normal conditions and less rapidly under latent conditions as compared to the control groups.

3) Differences between emotionality scores for pre-operative and 1st post-operative ratings and between pre-operative and 2nd post-operative ratings were significant.

4) There were no significant differences between groups in terms of food consumption.

CHAPTER IV

DISCUSSION

Consideration of the Experimental hypotheses.

A. The prediction that amygdalectomized animals would show significant attenuation of response in latency and running speed to shifts in magnitude of incentive was confirmed for the former but not the latter measure. The attenuation of latencies suggested by Figures 1 and 2 is verified by significant interactions for groups by incentive shift (Tables 1 and 2). No such significance was found for measures of running speed, however, although a comparison of the 1st and 6th post-shift sessions did indicate an attenuation effect for both amygdaloid and cortical lesions as compared with the unoperated controls (Figures 3 and 4).

Further data suggesting an attenuation of latency of response to shift conditions in the amygdalectomized group was provided by the initial and terminal post-shift sessions measures summarized in Figures 1 and 2. Although a comparison of latencies for these sessions indicated that response attenuation of the groups did not differ significantly, the data did suggest a definite trend of greater attenuation for amygdalectomized Ss.

B. A significant attenuation of latency and the lack of significant

attenuation of running speed to incentive shift would suggest that amygdalectomy disrupts the initiation more than the maintenance of behavior. Since latency responses were farther removed in time from the goal response than was running in the alley, incentive-motivation should have been more of a determining factor in the latter behavior. Moreover, the effect of incentive-motivation on running behavior should have been enhanced by the fact that the start box was gray, whereas both the runway and goal box were painted black. Thus, the statistical significance of latency attenuation coupled with the lack of significance of the running speed attenuation strongly suggested that the deficit was not primarily of a motivational nature.

A motivational point of view was previously rejected as an explanation of the response to incentive shift demonstrated by amygdalectomized monkeys in an operant situation (Schwartzbaum, 1960). The alternative suggested by Schwartzbaum was loss of ability to interrelate different reinforcing events or to respond to one set of events in terms of preceding sets, an interpretation which appears to complement a sequencing conception of limbic system function. This interpretation would also seem to be appropriate for the runway data of the present study.

The subnormal running speed of amygdalectomized Ss with a large incentive prior to shift (Figure 3) could have reflected a motivational deficit. It is quite possible, however, that it was the result of a depressed

physiological limit, since a similar comparative performance level was evident for latency measures (Figure 1). Histological data indicated, moreover, that this group sustained more amygdaloid damage than did the amygdalectomized Ss trained with a small incentive.

C. The lack of any significant group differences in acquisition errors on the T maze (Table 11) is compatible with a number of previous studies which have indicated that amygdalectomy does not significantly impair performance on standard instrumental discrimination tasks (Pribram and Bagshaw, 1953; Schwartzbaum and Pribram, 1960; Schwartzbaum, 1965). Other studies, however, have produced results suggesting that definite acquisition deficits do occur after amygdalectomy when positive incentives are used (Schwartzbaum, Thompson, Kellicutt, 1964; Schwartzbaum and Poulos, 1965). Moreover, Horvath (1963), using a negative incentive with cats, found that the retardation effects of amygdalectomy on acquisition increased noticeably as a function of complexity or difficulty of the task. It would seem, therefore, that the failure to find T maze acquisition differences between groups in the present study might be related to the low level of difficulty of the task. The ease with which Ss in all three groups appeared to master the task would certainly suggest that it was not sufficiently difficult as a discrimination learning task.

Although neither the T maze extinction conditions nor the groups by

conditions interaction was significant, a comparison of the means for this data suggested that the amygdalectomized Ss were unresponsive to the latent extinction procedure. This group, moreover, tended to extinguish more rapidly than the other two under normal extinction conditions. This more rapid extinction under normal conditions and less rapid extinction under latent conditions might suggest the loss of some motivational factor such as incentive-motivation. In fact, the observation that the amygdalectomized Ss required approximately the same number of trials to extinguish under latent as under normal conditions would seem to be explicable only by reference to a motivational construct. It is possible, however, that the normal extinction measures reflected non-significant associative rather than motivational differences in terms of acquisition.

Emotionality. Although the increase in emotionality observed in the present experiment (Figure 6) did not parallel the findings of many other studies, it nevertheless agreed with the results of several studies previously mentioned (Bard and Mountcastle, 1948; Schreiner and Kling, 1953). An explanation frequently advanced to explain instances of hyper-emotionality following amygdalectomy is that the lesions invaded the ventral hippocampus or in some way altered its activity. The present study, however, yielded inconclusive data concerning this hypothesis, since the rank order correlation of .25 between emotionality scores and

posterior extent of amygdaloid lesions did not approach statistical significance.

The fact that emotionality scores for several of the amygdalectomized animals increased from the 1st post-operative to the 2nd post-operative rating suggests a time course change following these lesions. Furthermore, this heightened emotionality did not appear to be reduced to any large extent by handling. In fact, the category contributing most to total emotionality score for these Ss was "resistance to handling."

Although the lesion effect on emotionality measures was statistically significant, the histological data seemed to present no readily specifiable neurological differentiation for this effect. The fact remains, however, that the amygdalectomized Ss showed, without exception, increases in emotionality scores from the pre-operative rating to the 1st post-operative rating. In addition, the final rating, taken after days of handling, showed slight decreases over the pre-operative ratings for only three animals (Appendix D, Table 19).

Food consumption. In the present study, no significant differences were found between groups in amount of food consumed. Although other investigators (Anand and Brobeck, 1952) found that amygdalectomy did not produce hyperphagia, Goddard (1964) indicated the most consistent picture of amygdaloid removal as being a transient decrease in food intake followed by longer lasting hyperphagia. Schwartzbaum (1960)

found that amygdalectomy did not increase hunger or drive for food, so the hyperphagia sometimes produced by these lesions would seem to reflect something other than enhanced motivational level. Schwartzbaum suggests that it is a general disturbance in habituation processes extending to classes of stimuli other than food.

The present failure to find any significant effects of lesions on food consumption is possibly due to an inadequate time lapse between the operations and the measures of food consumed. As mentioned previously, several of the experimental animals had to be force fed for a period of time after removal of their amygdala. Therefore, it is possible that eating habits had not been fully reestablished by the 11th post-operative day at which time the initial food consumption measures were taken. This early disruption of feeding behavior is further reflected in an average post-operative weight gain of 8.9 gm. for the amygdaloid lesion group as compared to gains of 20.6 and 16.6 gm. during the same time period for the cortical lesion and unoperated control Ss.

Synthesis. The significant data obtained from the runway experiment tended, in general, to support a behavioral sequencing interpretation of the amygdala and limbic system. A finding of significant latency attenuation plus a lack of such attenuation for running speeds on the 1st post-shift session seemed to negate an explanation in terms of incentive-motivation loss. A comparison of changes in running speed between the

initial and the terminal post-shift sessions did indicate, however, a significant attenuation effect of both amygdaloid and cortical lesions.

Hence, the effect as exhibited by running speed would seem to reflect non-specific brain damage.

Additional support for an attenuation effect applying exclusively to amygdalectomized Ss was provided through a comparison of initial and terminal post-shift latencies. Figures 1 and 2 indicated that Ss with cortical lesions did not show the same attenuation effect as did the amygdalectomized Ss.

Equivocal support for a motivational interpretation of amygdaloid function was provided by the T maze data, which suggested a decrease in resistance to normal extinction and a lack of responsiveness to latent extinction conditions for amygdalectomized Ss. Compared to the operated and unoperated controls, the Ss with amygdaloid lesions extinguished more slowly under latent conditions but more rapidly under normal conditions. Although the more rapid extinction under normal conditions could reflect an associative factor, the lack of responsiveness to latent extinction conditions seems to be most compatible with a motivational explanation. This explanation, however, would be in terms of reduced motivation, whereas the emotionality increases effected by these lesions might prompt exactly the opposite prediction. On the other hand, there is perhaps little justifiable rationale for expecting a significant relationship

between the decrease in reactivity threshold as measured by the King scale and an increase in sustained motivational level.

The food consumption data did not offer any support for a motivational interpretation of amygdaloid function. Perhaps the lack of significant results obtained for this variable was related to the relatively short time period elapsing between the operations and measures of food consumption.

CHAPTER V

SUMMARY

This study investigated the effect of amygdaloid and cortical lesions on the performance of laboratory rats in a straight runway with shifts in incentive magnitude and on their acquisition and latent extinction scores in a T maze. The lesion effects were also evaluated in terms of emotionality changes and amount of food consumed. The results indicated:

1. A significant attenuation of shift in starting latency to both increase and decrease in incentive magnitude that appeared to be primarily related to the Ss inability to interrelate different reinforcing events.

2. A lack of significant attenuation of change in running speed for the 1st session after incentive shift suggesting a negligible disruption of incentive-motivation.

3. A significant post-shift sessions effect on running speed, pointing to an attenuation for the cortical lesioned as well as the amygdalec-tomized Ss. This attenuation as reflected in the running speed seemingly represented a non-localized brain damage effect.

4. A lack of significant differences between groups in acquisition scores on the T maze possibly related to the low difficulty level of the task.

5. A non-significant decrease in resistance to extinction of amygdalectomized Ss under normal conditions which could reflect either a motivation or an association effect of the lesion. The lack of responsiveness of the amygdalectomized animals to latent extinction conditions would seem to be most compatible with a motivational interpretation.

6. Significant increases in emotionality produced by amygdalectomy that were apparently unrelated to any general increase in drive level.

7. A similarity in quantity of food consumed by amygdalectomized and control Ss, perhaps reflecting short term rather than the final effects of amygdaloid lesions on food intake.

The runway data was viewed as supporting a sequencing interpretation of limbic system function, whereas the T maze data, although not statistically significant, was most compatible with a motivation point of view.

APPENDICES

APPENDIX A

SIX RATING SCALES FOR THE STUDY OF EMOTIONALITY

- I. Reaction to Object Presentation: Pencil is presented close to animal's snout.
- 0--Rat ignores pencil
 - 1--Rat alert and attentive, some body tenseness
 - 2--Legs and body tense and immobile, vibrissae point forward
 - 3--Scurries away or makes an occasional mild biting attack on pencil
 - 4--Intermediate
 - 5--Very aggressive attack, disorganized panic, or violent flight
- II. Response to Tap on Back
- 0--No reaction
 - 1--Twitching or restlessness
 - 2--Twitching or scurrying away
 - 3--Jumps or hops up a bit, but then settles down
 - 4--Leaps in air and runs about in fright. Big hop and movement after
 - 5--Leaps violently, dashes off in panic, frantic rebounding from side to side of cage.
- III. Resistance to Capture: Glove is extended forward to animal slowly and rat is grasped firmly but not roughly.
- 0--Remains calm, does not move when approached, does not struggle when grasped
 - 1--Remains calm when approached, but scurries away and tugs a bit when grasped
 - 2--Retreat and moderate struggle
 - 3--Retreats when approached, struggles vigorously when grasped
 - 4--Strong attempt to escape when approached, struggles strongly and is disorganized. Some biting
 - 5--Leaps violently when grasped, bites frantically, exceedingly difficult to catch
- IV. Resistance to Handling: Animal passed from hand to hand for about 30 seconds.
- 0--Relaxes in hand, does not attempt to escape
 - 1--Restless with some feeble squirming
 - 2--Sporadic attempts to pull out of hand
 - 3--Struggles pretty continuously and quite virorous in attempt to escape
 - 4--Bites also
 - 5--Frantic biting, powerful tugging and disorganized twisting

V. Vocalization to Capture and Handling

0--None

1--Few squeaks

2--Frequent squeaking

3--Frequent squealing-squawking

4--Squealing continuous

5--Frantic and loud screeching continued

VI. Urination and Defecation in Reaction to Handling

0--None

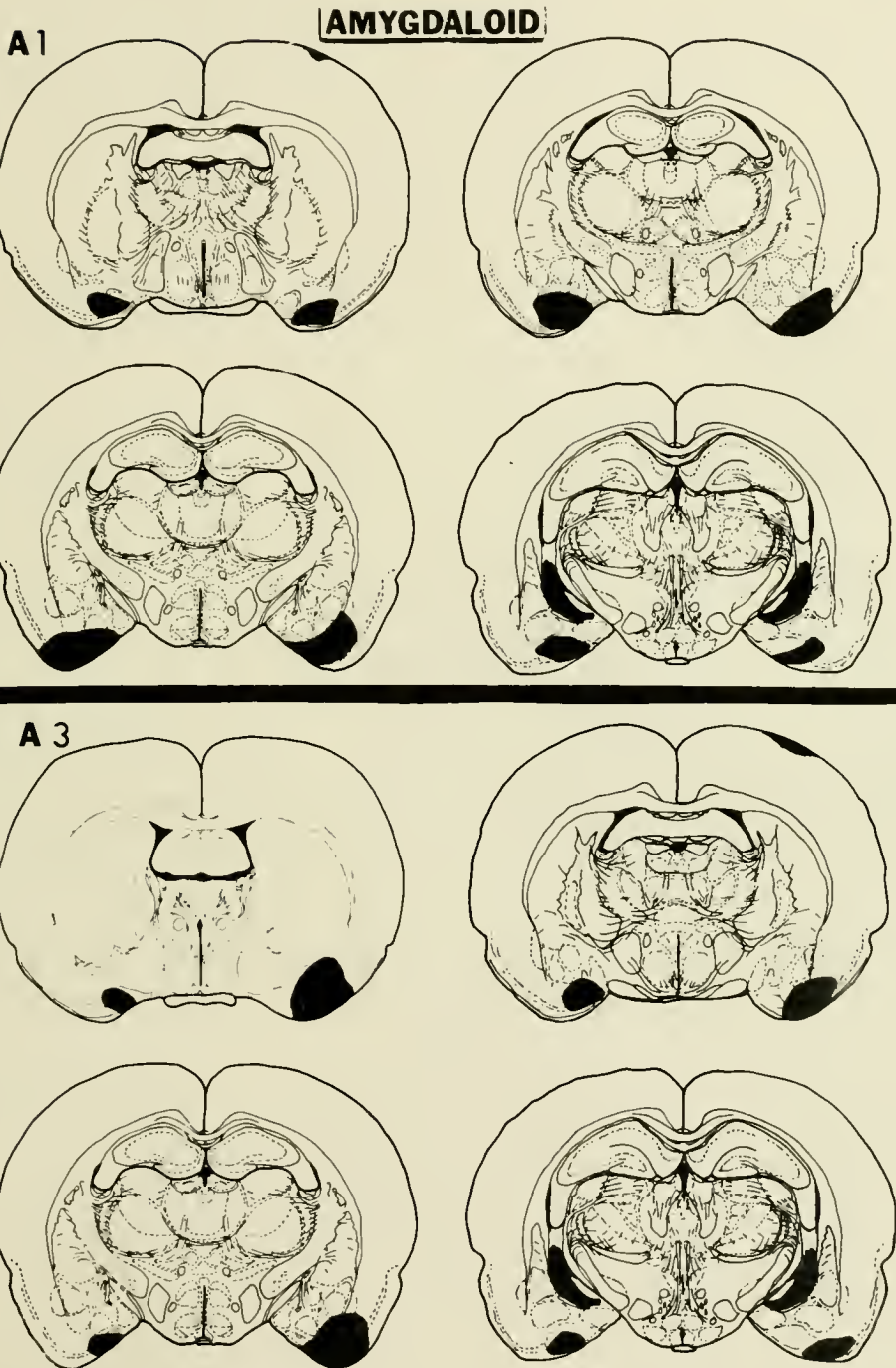
1--Slight urination

2--Few stools

3--Loose stools

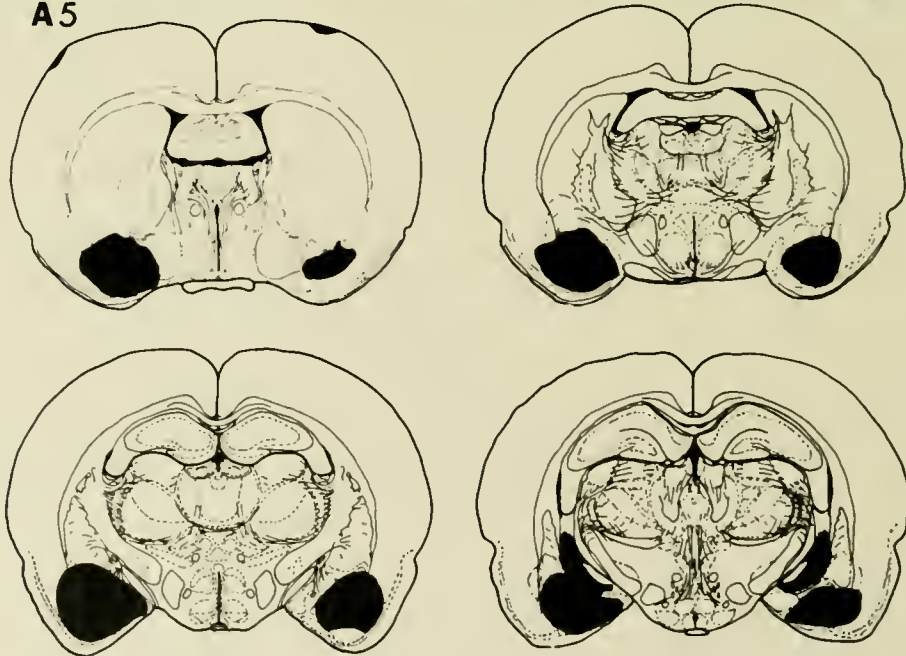
APPENDIX B
FRONTAL BRAIN SECTIONS

Animals 1 and 3



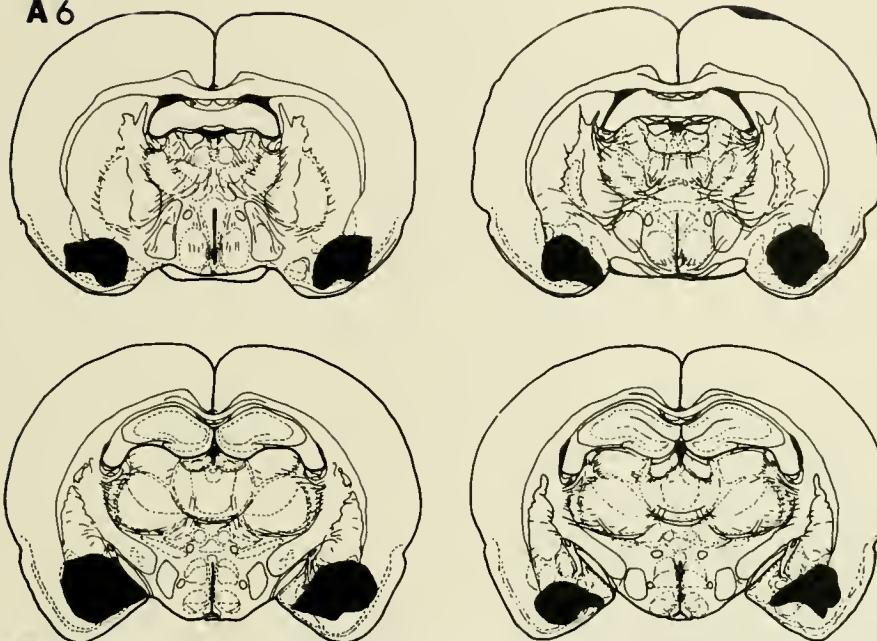
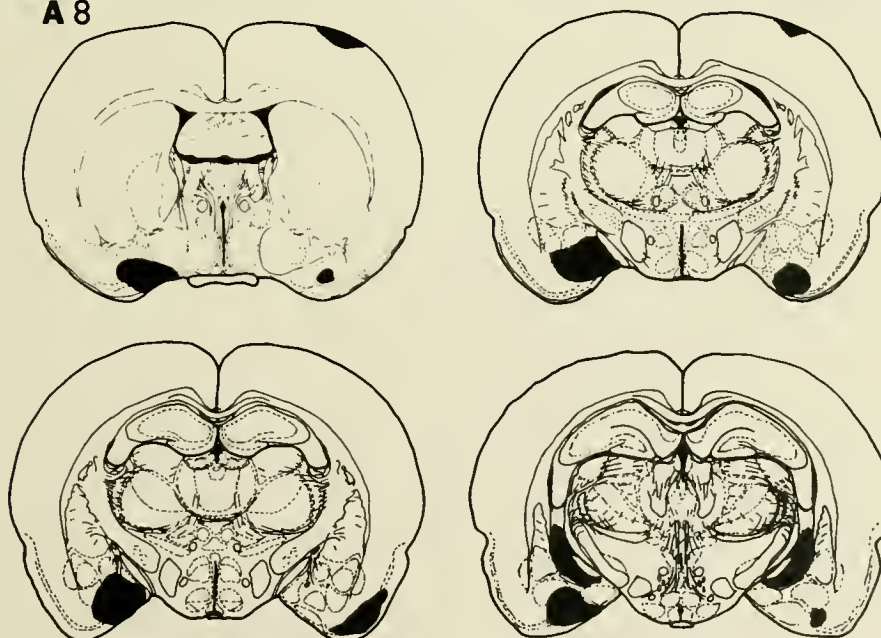
FRONTAL BRAIN SECTIONS

Animals 4 and 5

A 4**A 5**

FRONTAL BRAIN SECTIONS

Animals 6 and 8

A6**A8**

FRONTAL BRAIN SECTIONS

Animals 21 and 22

A 21



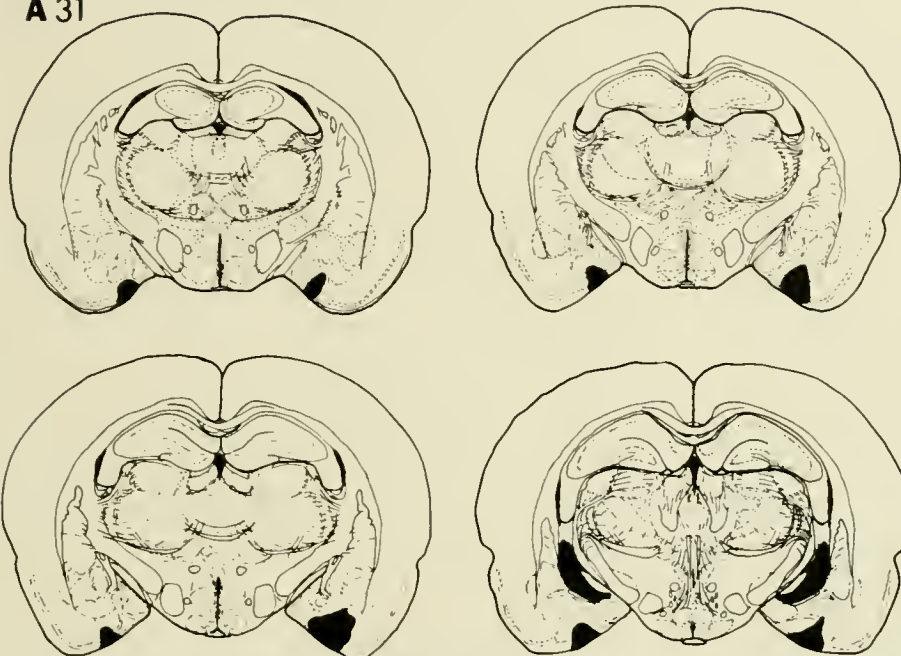
A 22



FRONTAL BRAIN SECTIONS

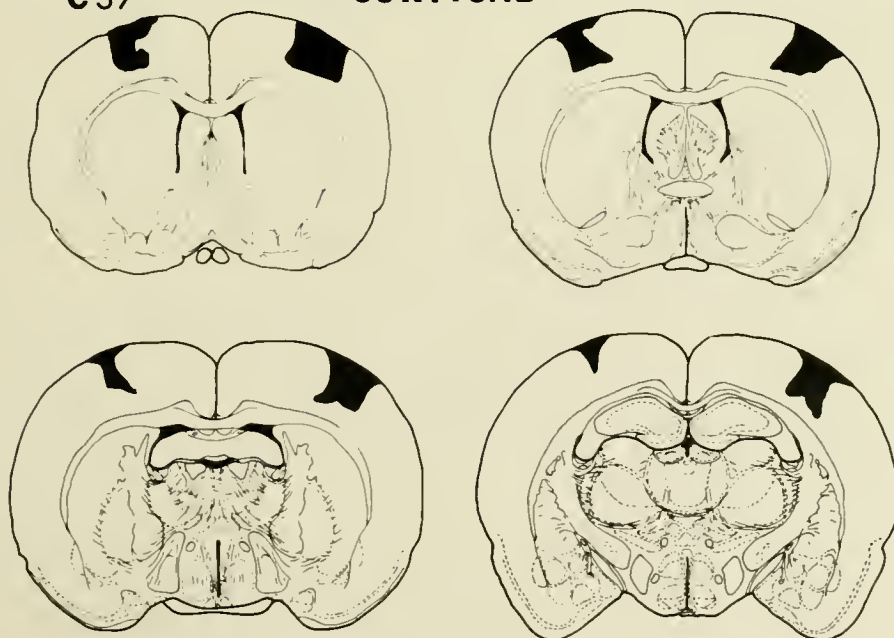
Animals 31 and 37

A 31



C 37

CORTICAL



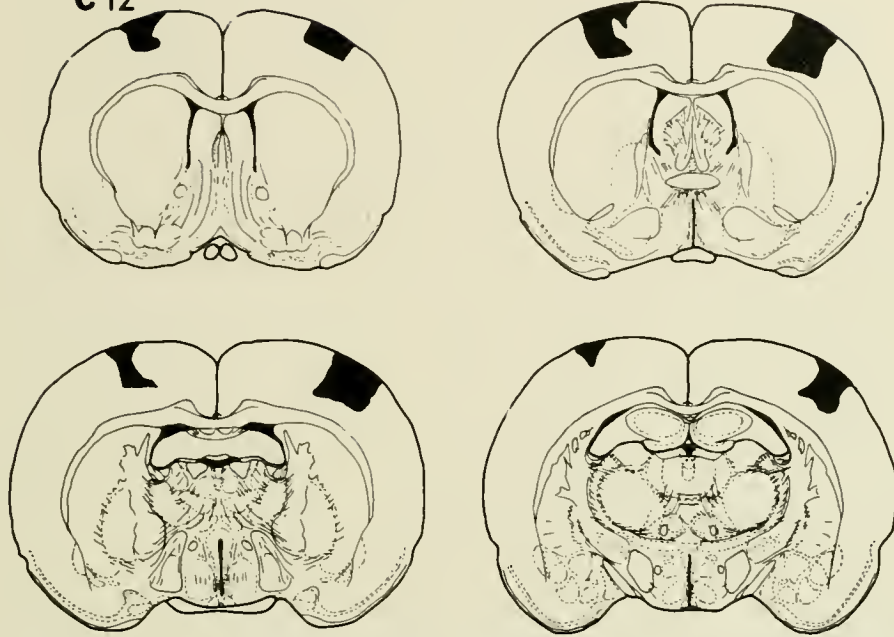
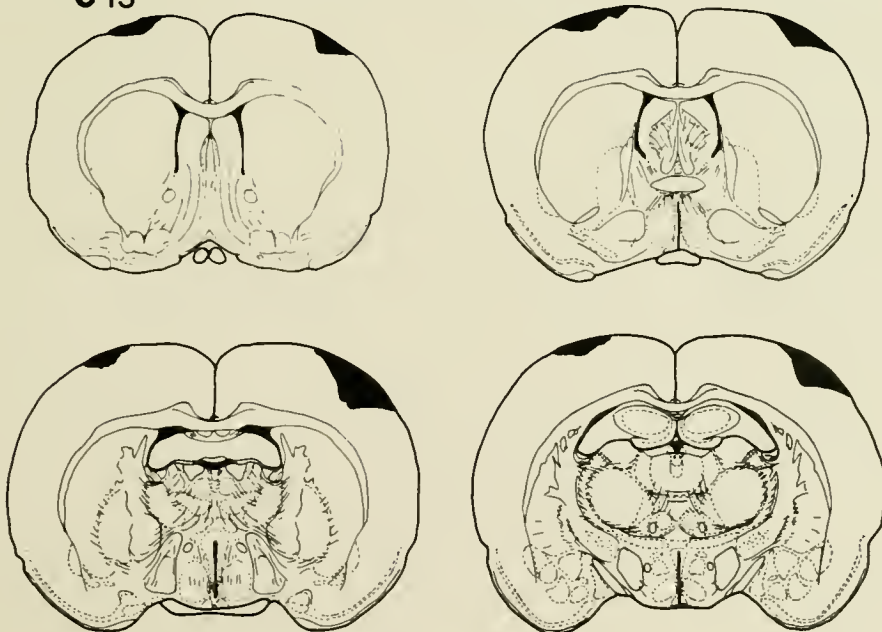
FRONTAL BRAIN SECTIONS

Animals 10 and 11

C 10**C 11**

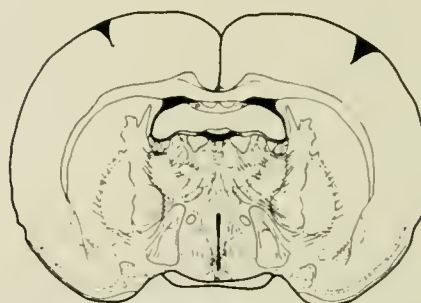
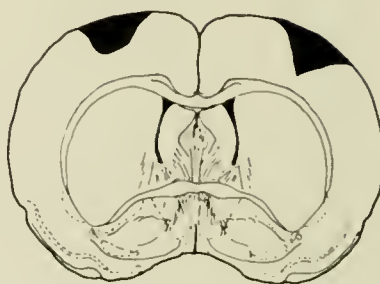
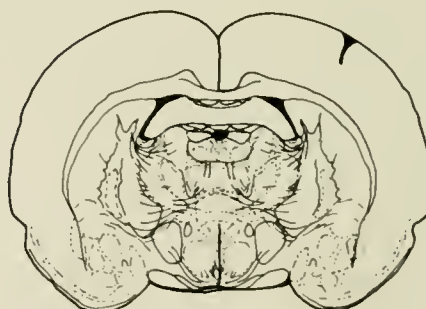
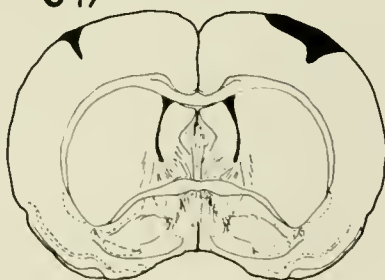
FRONTAL BRAIN SECTIONS

Animals 12 and 13

C 12**C 13**

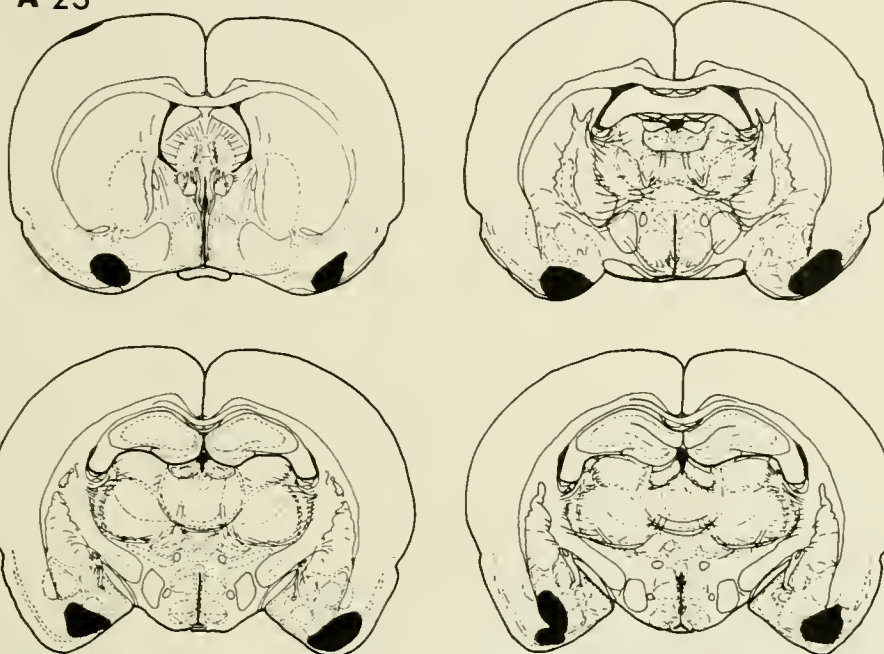
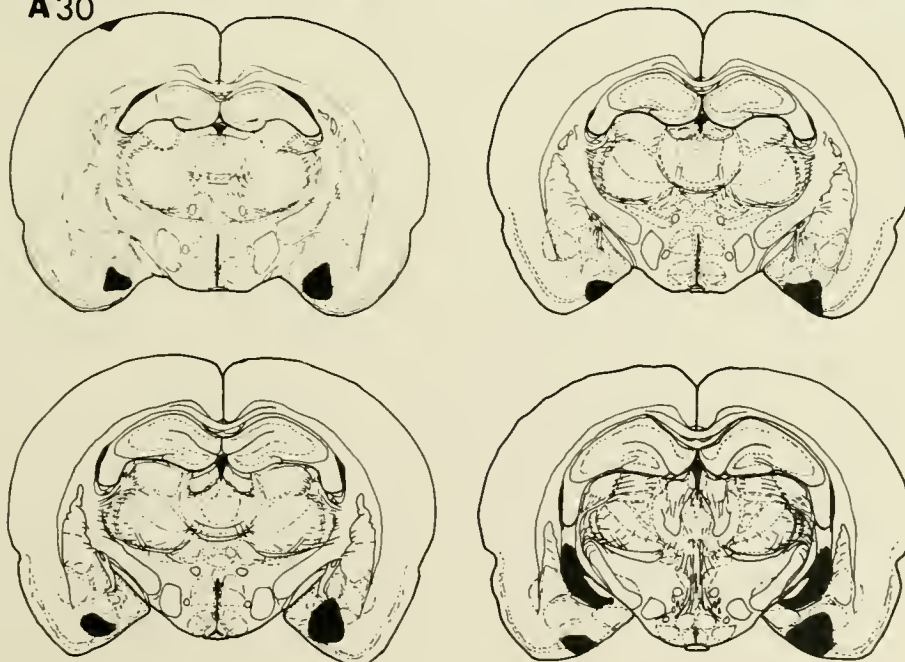
FRONTAL BRAIN SECTIONS

Animals 14 and 17

C14**C17**

FRONTAL BRAIN SECTIONS

Animals 23 and 30

A 23**A 30**

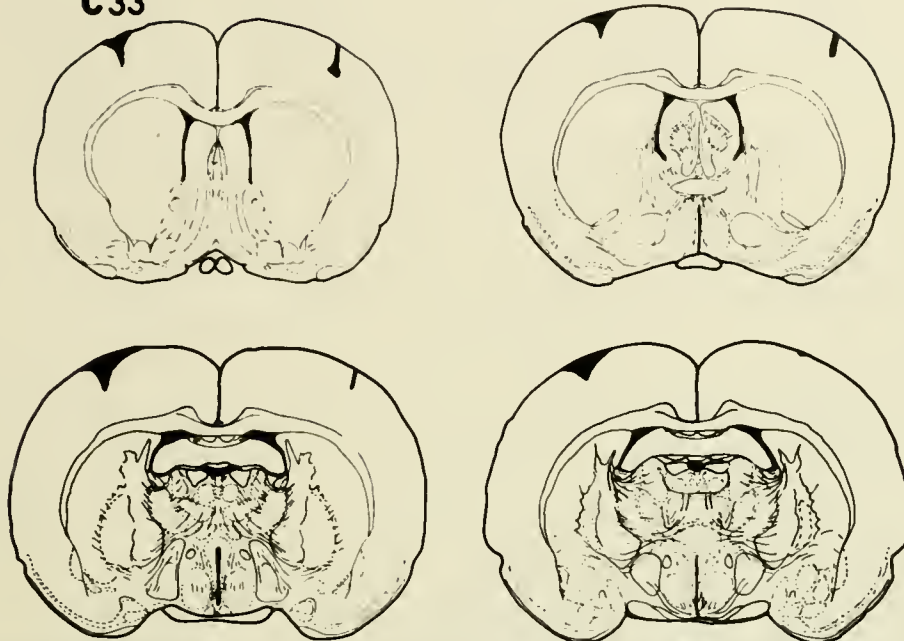
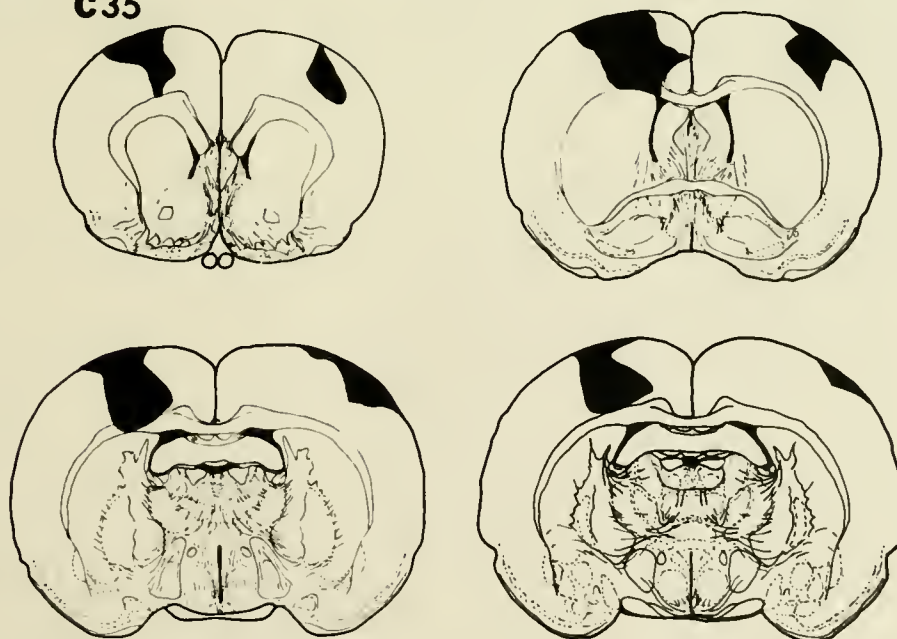
FRONTAL BRAIN SECTIONS

Animals 24 and 26

C24**C26**

FRONTAL BRAIN SECTIONS

Animals 33 and 35

C33**C35**

APPENDIX C

TABLE 14

MEANS OF RECIPROCAL OF STARTING LATENCY FOR LAST
PRE-SHIFT SESSION WITH 90 MG. REWARD AND 1ST AND
6TH POST-SHIFT SESSIONS WITH 945 MG. REWARD

Animal Number	Last Pre-Shift	1st Post-Shift	6th Post-Shift
Amygdaloid Lesions			
21	2.13	4.29	5.13
22	3.00	5.55	4.37
23	1.17	.93	4.32
30	2.67	2.74	4.21
31	2.93	2.74	4.60
	<u>11.90</u>	<u>16.25</u>	<u>22.63</u>
Cortical Lesions			
24	4.18	4.55	4.76
26	.38	3.27	4.59
33	2.85	4.66	4.72
35	1.53	2.77	3.15
37	2.33	3.07	2.88
	<u>11.27</u>	<u>18.32</u>	<u>20.10</u>
Unoperated Controls			
27	3.89	5.54	4.91
28	1.61	4.21	5.07
29	2.51	5.26	5.65
36	3.12	8.97	7.52
38	2.75	5.52	4.98
40	3.12	5.37	6.54
	<u>17.00</u>	<u>34.87</u>	<u>34.67</u>
Sum of Sums	40.17	69.44	77.40

TABLE 15

MEANS OF RECIPROCAL OF STARTING LATENCY FOR LAST
PRE-SHIFT SESSION WITH 945 MG. REWARD AND 1ST AND
6TH POST-SHIFT SESSIONS WITH 90 MG. REWARD

Animal Number	Last Pre-Shift	1st Post-Shift	6th Post-Shift
Amygdaloid Lesions			
1	1.55	3.82	2.08
5	2.23	2.76	2.80
8	5.02	5.07	1.30
3	6.93	3.67	2.95
4	4.46	5.37	2.30
6	3.76	1.54	1.43
	<u>23.95</u>	<u>22.23</u>	<u>12.86</u>
Cortical Lesions			
11	2.93	1.73	2.61
14	5.41	6.98	6.94
17	4.56	2.30	.83
10	5.94	3.30	2.68
12	5.12	6.01	5.35
13	6.45	4.58	3.64
	<u>30.41</u>	<u>24.90</u>	<u>22.05</u>
Unoperated Controls			
2	8.15	1.49	6.38
7	6.02	4.50	2.84
9	8.64	4.47	1.71
15	5.50	2.73	2.24
16	4.59	3.03	3.26
18	5.37	2.78	1.95
	<u>38.27</u>	<u>19.00</u>	<u>18.38</u>
Sum of Sums	92.63	66.13	53.29

TABLE 16

MEANS OF RUNNING SPEED FOR LAST PRE-SHIFT SESSION
WITH 90 MG. REWARD AND 1ST AND 6TH POST-SHIFT
SESSIONS WITH 945 MG. REWARD

Animal Number	Last Pre-Shift	1st Post-Shift	6th Post-Shift
Amygdaloid Lesions			
21	.792	1.007	1.005
22	.902	1.057	1.030
23	.610	.598	.718
30	.590	.831	.961
31	.654	.804	.814
	<u>3.548</u>	<u>4.297</u>	<u>4.528</u>
Cortical Lesions			
24	.870	.964	.989
26	.479	.855	.857
33	.837	1.204	.814
35	.722	.760	.892
37	.587	.802	.777
	<u>3.495</u>	<u>4.585</u>	<u>4.329</u>
Unoperated Controls			
27	.670	.816	.895
28	.583	.788	1.027
29	.666	.863	.924
36	.740	.974	1.182
38	.687	.809	.924
40	.581	.894	1.165
	<u>3.927</u>	<u>5.144</u>	<u>6.117</u>
Sum of Sums	10.970	14.026	14.974

TABLE 17

MEANS OF RUNNING SPEED FOR LAST PRE-SHIFT SESSION
WITH 945 MG. REWARD AND 1ST AND 6TH POST-SHIFT
SESSIONS WITH 90 MG. REWARD

Animal Number	Last Pre-Shift	1st Post-Shift	6th Post-Shift
Amygdaloid Lesions			
1	.374	.227	.605
5	.417	.210	.790
8	.693	.328	.632
3	.738	.256	.655
4	.669	.110	.415
6	.672	.247	.072
	<u>3.563</u>	<u>1.378</u>	<u>3.169</u>
Cortical Lesions			
11	.843	.357	.703
14	.824	.419	.713
17	.812	.308	.619
10	1.042	.233	.777
12	1.076	.742	.943
13	.725	.313	.359
	<u>5.322</u>	<u>2.372</u>	<u>4.114</u>
Unoperated Controls			
2	.814	.707	.441
7	.963	.168	.253
9	1.290	.330	.503
15	.755	.548	.521
16	.749	.614	.685
18	.743	.792	.616
	<u>5.314</u>	<u>3.159</u>	<u>3.019</u>
Sum of Sums	14.199	6.909	10.302

TABLE 18

RECIPROCAL OF RUNNING TIME BY TRIALS
FOR 1ST POST-SHIFT SESSION

Trials			
1st	2nd	3rd	4th
Amygdaloid Lesions			
.185	.272	.279	.172
.200	.129	.084	.429
.671	.411	.027	.204
.521	.211	.225	.068
.267	.056	.085	.032
.337	.417	.195	.040
<u>2.181</u>	<u>1.496</u>	<u>.895</u>	<u>.945</u>
Cortical Lesions			
.610	.274	.127	.417
.869	.435	.274	.099
.704	.050	.227	.252
.153	.308	.32	.339
.980	.980	.909	.098
.621	.057	.267	.308
<u>3.937</u>	<u>2.104</u>	<u>1.936</u>	<u>1.513</u>
Unoperated Controls			
.893	.847	.094	.360
.351	.901	.372	.833
1.219	1.000	.909	.042
.135	.134	.021	.035
.575	.034	.025	.037
.461	.617	.114	.128
<u>3.634</u>	<u>3.533</u>	<u>1.535</u>	<u>1.435</u>
Sum of Sums			
9.752	7.133	4.366	3.893

APPENDIX D

TABLE 19

MEAN EMOTIONALITY SCORES FOR PRE-OPERATIVE
RATING AND TWO POST-OPERATIVE RATINGS

Animal Number	Pre-Operative	Post-Operative Pre-Training	Post-Operative Post-Training
Amygdaloid Lesions			
1	6.25	9.33	6.78
5	10.35	11.58	13.65
8	10.35	10.67	9.90
3	6.05	7.58	8.25
4	7.10	8.08	7.40
6	9.45	10.41	9.60
21	9.00	10.08	9.75
22	8.40	10.58	6.65
23	6.50	11.00	10.60
30	6.95	10.25	7.25
31	8.65	9.75	5.95
Cortical Lesions			
11	7.10	7.83	8.00
14	8.25	6.17	8.80
17	8.85	5.92	5.00
10	8.50	7.08	6.50
12	6.25	6.92	7.05
13	11.25	13.25	9.50
24	7.35	10.25	6.80
26	5.10	6.50	5.45
33	8.30	6.83	7.25
35	8.25	7.33	6.65
37	9.10	11.08	died

APPENDIX E

TABLE 20

RANK ORDER OF POSTERIOR EXTENT OF AMYGDALOID
LESIONS AND ANTERIOR-POSTERIOR EXTENT IN MICRA

Animal Number	Rank of Posterior Extent	Anterior-Posterior Extent
5	1.5	1900
6	1.5	2700
4	3.0	2400
8	4.0	2100
3	5.0	2100
1	7.5	1600
22	7.5	2000
30	7.5	500
31	7.5	800
23	10.0	1400
21	11.0	1200

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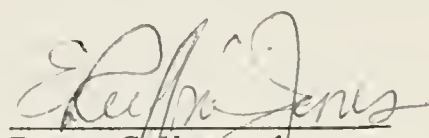
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BIOGRAPHICAL SKETCH

Wilbur Alan Thomas was born July 31, 1929 in LaBelle, Florida. He was graduated from LaBelle High School in 1947, and served from 1951 until 1953 in the Armed Forces of the United States. He received his B.S. and M.A. degrees from the University of Florida in 1955 and 1957. He was employed as a psychologist by the Georgia State Institution for Mentally Retarded at Gracewood from 1958 to 1961. He returned to graduate school in 1961 and pursued graduate studies until the fall of 1963. From 1963 to the present he has been employed as assistant professor of psychology by Rollins College in Winter Park, Florida. He is married to the former Miss Frances Marie Glover and they have four children.

This dissertation was prepared under the direction of the chairman of the candidate's supervisory committee and has been approved by all members of that committee. It was submitted to the Dean of the College of Arts and Sciences and to the Graduate Council, and was approved as partial fulfillment of the requirements for the degree of Doctor of Philosophy.

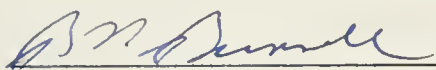
December 17, 1966



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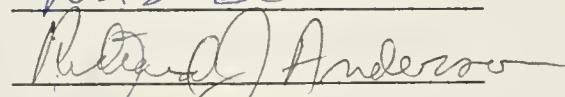
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